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LETTER TO THE EDITOR

Human carbofuran intoxication with myocardial injury mimicking acute myocardial infarction

A 51-year-old man with a history of hypertension drank about 50 mL of pesticide, containing carbofuran, after an argument with his family. He was found drowsy, weak, and unable to talk about 15 minutes after the argument. No chest tightness, diaphoresis, dyspnea, or chest pain were noted. He was brought to the hospital immediately, presenting with tachycardia (112 beats/min), hypertension (192/96 mmHg), and tachypnea (26 breaths/min). Physical examination revealed drowsy and agitated consciousness (Glasgow coma scale of E3V2M5) and small pupil size, but no obvious airway secretion or abnormality in breath sound, heart sound or abdomen. The laboratory examination revealed decreased cholinesterase level (3.67 kU/L). Electrocardiography showed diffuse ST-T segments depression over precordial leads (Fig. 1A). Gastric lavage with activated charcoal and atropine infusion were given. He developed hypoxemic respiratory failure 2 hours later, requiring intubation for mechanical ventilation. After admission to the intensive care unit, adequate hydration and atropine infusion were prescribed for suspected carbofuran intoxication. Elevated cardiac enzyme levels (creatine phosphokinase, 242 IU/L; creatine kinase-MB, 15.7 ng/mL; and troponin-I, 2.04 ng/mL) were noted, so dual antiplatelets and heparinization were given because acute myocardial infarction (AMI) could not be excluded. However, the cardiac enzyme levels reached a peak within a few hours, and the follow-up electrocardiography showed interval disappearance of the ST-T segments depression (Fig. 1B). Bedside echocardiography showed adequate systolic function without valvular abnormality or wall motion defect. Tachyarrhythmia-related injury was less likely because tachycardia soon subsided at the emergency department. Stress cardiomyopathy was also not favored because echocardiography showed normal wall motion. Therefore, carbofuran-related cardiotoxicity, rather than AMI, tachyarrhythmia or stress cardiomyopathy, was concluded, so clopidogrel and heparin were discontinued. Cardiac catheterization was suggested but refused because of no further associated cardiac symptoms and signs. He was soon discharged uneventfully without obvious sequelae in 1 week.

Carbofuran is one of the most potent and toxic carbamate pesticides. Its toxic effects are mainly due to its inhibition on cholinesterases. Besides its neurotoxicity, it could also induce hypercontraction of skeletal muscles, resulting in skeletal myotoxicity [1]. Carbofuran is also a notorious endocrine disrupting agent, which may interfere with the function of many hormones, resulting in infertility [1]. Other reported toxic effects include blurred vision, coma, nausea, sweating, acute respiratory distress syndrome, cortical blindness, and acute pancreatitis [2,3]. The severity of clinical features may be associated with inhibition of cholinesterase activity in the blood, which is usually rapidly reversible. In most cases, complete recovery occurs within 2–3 hours, with or without atropine treatment. Therefore, occupational acute carbofuran poisoning is usually characterized by rapid onset, mild illness with quick recovery.

Myocardial injury related to carbofuran in humans has been seldom reported, although it is a well-known cardiotoxic compound in rats. Its toxicity may be associated with the release of nonphysiological amounts of endogenous catecholamines. A recent epidemiological study revealed that carbofuran exposure may increase the risk of myocardial infarction among farmwomen, while the precise mechanism remained elusive [4]. Because of its profound toxicity, carbofuran is banned in many countries. Its intoxication, especially cardiotoxicity is even rarely reported.

In conclusion, we present a rare case of carbofuran intoxication with myocardial injury mimicking AMI. Clinicians should be alert about the possible cardiotoxicity related to pesticide intoxication.

Conflicts of interest: All authors declare no conflicts of interest.

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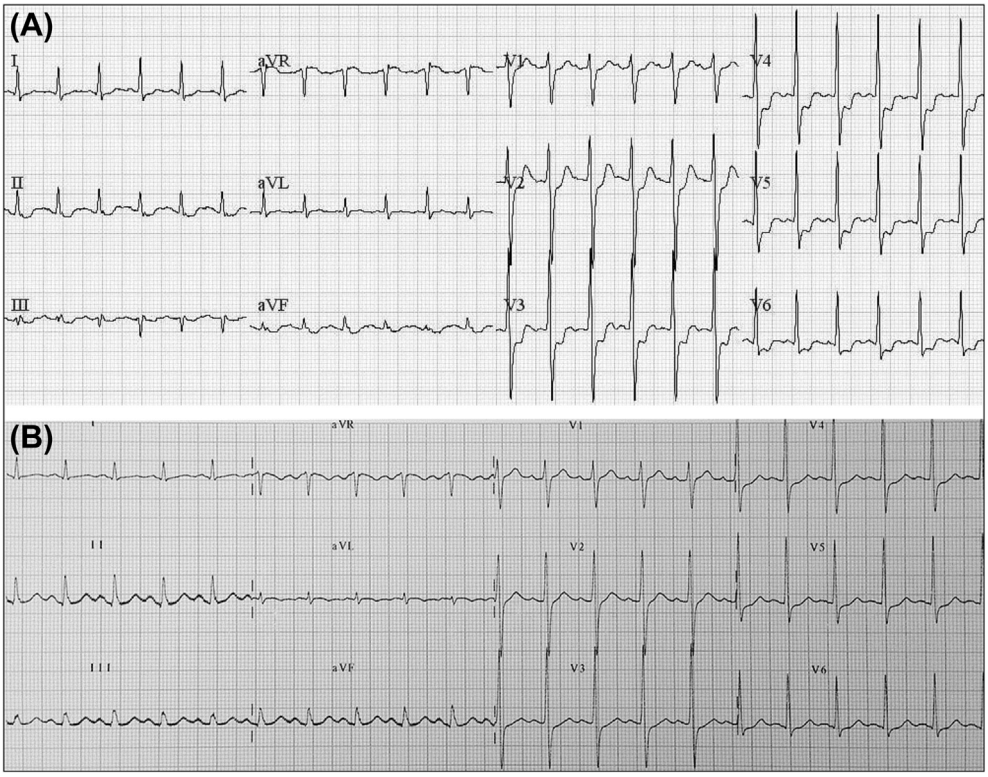

Figure 1. (A) Electrocardiography on presentation revealed diffuse ST segment depression in the precordial leads. (B) The follow-up electrocardiography performed 10 hours later shows interval resolution of the ST segment depression.

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